

EDITORIAL

Oilseed rape – allergen or irritant?

Oil has been produced from oilseed rape and other *Brassica* seed crops in Europe since at least the 15th Century [1]. In the 17th Century, sufficient seed was produced in the UK to meet home demand and leave a surplus for export. Production then gradually declined in the face of competition from imports until the acreage dropped to about 2000 hectares in the mid 1960s. Not until recently has the full potential of the crop been realized. During the past 20 years there has been a steady increase in the acreage of the crop harvested reaching a peak in 1993 with 377 000 hectares being grown. This represents 6% of the total crop area [2].

In 1954, Colldahl [3] reported a case of asthma and pollinosis which proved to be due to hypersensitivity to rape pollen. Since then, a number of other studies carried out in areas of high oilseed rape (OSR) cultivation have supported the suggestion that the crop causes allergy. Bucur and Arner [4] showed that 23% of allergic patients in different parts of Sweden where OSR was grown showed positive skin tests to OSR pollen. Parratt *et al.* [5] found 26.3% seropositivity to OSR pollen in rural Tayside in a study of serological responses.

The possibility that symptoms attributed to OSR are due to the presence of cross-reactive allergens in grass pollen has been suggested [6,7] and this topic is addressed by Welch and coworkers in this issue [8].

Charles Blackley [9] first showed that ‘hay-fever’ was caused by exposure to grass pollen. An Aberdeen-based study [10] found that grass pollen was the third most common allergen in atopic children. Welch *et al.* in this issue [8] have examined the hypothesis that sensitization to OSR was due to cross-reactivity with grass pollen. They demonstrated that individuals with occupational exposure to OSR were all sensitized to grass pollen. Western blotting showed that allergens of similar molecular weights were present in both types of pollen, suggesting common allergens existed. However, grass pollen was unable to inhibit the binding of immunoglobulin (Ig) E to OSR and *vice versa*, although there was complete self-inhibition. Welch *et al.* concluded therefore, that there was no evidence of any cross-reactivity between OSR and grass pollen.

There is little doubt that the oilseed rape crop can induce allergy [4,11]. There is also evidence that those in close contact with this crop have allergic disease [12]. However, in controlled population studies, it has not proved possible to relate symptoms to a particular component of the crop [13]. One explanation for this could be that no one component of the crop is to blame, rather symptoms of allergy to OSR are caused by two or more components acting either together or separately.

It is known that volatile organic compounds are emitted from OSR during flowering and these have been cited as possible aeroallergens [14] although their effects on the population is generally unknown.

Moulds growing on the OSR crop may also be responsible for some of the symptoms. Parratt *et al.* [5] found that RAST positivity for mixed moulds was as high as 17% and sensitivity to *Alternaria alternata*, which is commonly found on OSR, has been shown to be a risk factor for sudden, severe episodes of asthma [15].

Work carried out in our laboratory has shown that exposure to a common fungal parasite of OSR, *Botrytis cinerea*, occurs frequently in rural dwellers and that IgE sensitization occurs in a significant number of those exposed when susceptible OSR crop strains are cultivated [16]. Preseason measurement showed that 11.1% of participants had detectable IgE antibodies to this fungus and 36.5% were positive at the post-season recall ($P < 0.001$).

One further reason for the inability to relate symptoms caused by the OSR crop to one particular component of the crop could be that although the reaction to OSR seems superficially to manifest itself as true allergy (i.e. type 1 hypersensitivity), this may not always be the case. A large number of individuals who record symptoms of ‘allergy’ when in contact with this crop do not have IgE to either pollen or moulds [13]. Therefore, if the symptoms are not allergic, there must be some other reason for the effects of this crop.

A study carried out in Aberdeen [17] measured the levels of allergens and other potentially harmful substances released by the OSR crop. OSR was shown to give off terpenes which are simple cyclic organic chemicals, locally irritant to mucous membranes. These were detected close to fields. This – together with the fact that irritant-type symptoms, e.g. cough, headache, are usually experienced by OSR ‘allergy’ sufferers – suggests that the volatile organic compounds (VOCs) released by the flowering plant may be responsible for at least some of the symptoms experienced by individuals living in the vicinity of the OSR crop.

Butcher *et al.* [14] agreed with this hypothesis and made the point that since OSR is grown in dense monoculture, levels of these common VOCs are much greater in close proximity to the crop than would normally be experienced, perhaps sufficient to act as irritants or allergens.

Parratt *et al.* [13] showed that symptom reporting was at its highest coincidence with peak OSR flowering but did not correlate with levels of OSR pollen. They also demonstrated that reporting of irritant-type symptoms was high.

Another study from the Grampian area of Scotland concluded that subjects whose respiratory symptoms coincided with the OSR flowering season simultaneously develop an increase in bronchial reactivity and that this increase is mediated by factors other than IgE [18].

In the light of all the above evidence, it would appear that although there is a definite reaction to the OSR crop, not all of this is allergic and in the absence of atopy, individuals who exhibit symptoms and increased bronchial reactivity could be reacting to VOCs in the air.

Whatever the reason, the effects of this crop cannot be easily accounted for by common immunological explanations. Welch *et al.* have assisted the search for understanding by clearly showing that antigen cross-reaction between OSR and grass pollens is unlikely.

References

- 1 Fussell GE. History of cole (*Brassica* spp.). *Nature*, London 1955; 176:48–51.
- 2 Carruthers SP, Marsh JS, Turner PW *et al.* Industrial markers for UK-produced oilseeds. London: Home Growth Cereals Authority, 1995.
- 3 Colldahl H. Rape pollen allergy – report of a case. *Acta Allergol* 1954; 7:367–9.
- 4 Bucur I, Arner B. Rape pollen allergy. *Scand J Respir Dis* 1978; 59:222–7.
- 5 Parratt D, Thomson G, Saunders C, McSharry C, Cobb S. Oilseed rape as a potent allergen. *Lancet* 1990; 335:121–2.
- 6 Hemmer W. The health effects of oilseed rape: myth or reality? *Br Med J* 1998; 316:1327–8.
- 7 Hemmer W, Focke M, Wantke F, Jager S, Gotz M, Jarisch R. Oilseed rape is a potentially relevant allergen. *Clin Exp Allergy* 1997; 27:156–61.
- 8 Welch J, Jones MG, Cullinan P, Coates OA, Newman Taylor AJ. Sensitization to oilseed rape is not due to cross-reactivity with grass pollen. *Clin Exp Allergy* 2000; 30:370–5.
- 9 Blackley CH. Experimental researches on the causes and nature of catarrhus aestivus. Dawson Publishing Co. London, 1959:57–8 (Reprinted from Bailliere, Tindall and Cox, 1873).
- 10 Ninan TK, Russell G. Respiratory symptoms and atopy in Aberdeen schoolchildren: evidence from two surveys 25 years apart. *Br Med J* 1992; 304:873–5.
- 11 McSharry C. New aeroallergens in agriculture and related practice. *Clin Exp Allergy* 1992; 22:423–6.
- 12 Fell PJ, Soulsby S, Blight MM, Brostoff J. Oilseed rape – a new allergen? *Clin Exp Allergy* 1992; 22:501–5.
- 13 Parratt D, McFarlane Smith W, Thomson G, Cameron LA, Butcher RD. Evidence that Oilseed rape (*Brassica napus* ssp. *Oleifera*) causes respiratory illness in rural dwellers. *Scot Med J* 1995; 40:74–6.
- 14 Butcher RD, McFarlane Smith W, Robertson GW, Griffiths DW. The identification of potential aeroallergens from oilseed rape (*Brassica napus* ssp. *Oleifera*): volatile organic compounds emitted during flowering progression. *Clin Exp Allergy* 1994; 24:1105–14.
- 15 O'Halleren MT, Yunginger JW, Offord KP *et al.* Exposure to an aeroallergen as a possible precipitating factor in respiratory arrest in young patients with asthma. *New Eng J Med* 1991; 324:359–63.
- 16 Galloway DM. Antibody to *Brassica napus* (oilseed rape) and its Fungi in Patients with Allergy. MPhil Thesis, University of Abertay Dundee, 1996.
- 17 Souter A, Harker C, Seaton A, Brooke M, Marr I. OSR and seasonal symptoms: epidemiological and environmental studies. *Thorax* 1994; 49:352–6.
- 18 Packe GE, Soutar A, Harker C, Friend JAR, Seaton A. Bronchial reactivity and immunoglobulin E concentrations in subjects with respiratory symptoms associated with the flowering of oilseed rape. *Proc Br Thoracic Soc* 1992; 47:885.

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